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1: Cornell Vet. 1992 Jan;82(1):41-52. Related Articles, Links

Erratum in:
- Cornell Vet 1992 Apr;82(2):115.

Characterization of Sarcocystis neurona from a thoroughbred with equine protozoal myeloencephalitis.

Bowman DD, Cummings JF, Davis SW, deLahunta A, Dubey JP, Suter MM, Rowland PH, Conner DL.

Department of Microbiology, College of Veterinary Medicine, Cornell University, Ithaca, New York 14853.

Morphological information is presented for syntype material of the etiologic agent of equine protozoal myeloencephalitis, Sarcocystis neurona. A clinical description of the horse from which the organism was isolated and the methodology used to immunosuppress the horse in an attempt to increase parasite numbers are also given. The description includes microscopic details observed both with light and transmission electron microscopy. Mainly stages from tissue are illustrated, but information is also presented on the development of the organism after inoculation onto monolayers of bovine monocytes. It is believed that the large numbers of organisms observed in this horse were due to its having not received prior treatment with trimethoprimsulphonamide and the large amounts of corticosteroids that were administered in order to facilitate isolation of the pathogen.

Publication Types:
- Case Reports

PMID: 1740059 [PubMed - indexed for MEDLINE]

J Parasitol. 1995 Dec;81(6):916-9. Related Articles, Links

Identification of opossums (Didelphis virginiana) as the putative definitive host of Sarcocystis neurona.

Fenger CK, Granstrom DE, Langemeier JL, Stamper S, Donahue JM, Patterson JS, Gajadhar AA, Marteniuk JV, Xiaomin Z, Dubey JP.

Department of Veterinary Sciences, University of Kentucky, Lexington 40546, USA.

Sarcocystis neurona is an apicomplexan that causes equine protozoal myeloencephalitis (EPM) in North and South America. Horses appear to be an aberrant host, because the merozoites continually divide in the central nervous system, without encysting. The natural host species has not previously been identified. The small subunit ribosomal RNA (SSURNA) gene of S. neurona was compared to those of Sarcocystis muris, Sarcocystis cruzi, Toxoplasma gondii, and Cryptosporidium parvum to identify a unique region suitable for a species-specific amplification primer. The S. neurona SSURNA primer was used in a polymerase chain reaction (PCR) assay for the purpose of identifying this organism in feces and intestinal digest of wildlife specimens. Sporocysts were isolated from 4 raccoons (Procyon lotor), 2 opossums (Didelphis virginiana), 7 skunks (Mephitis mephitis), 6 cats (Felis catus), 1 hawk (Accipiter sp.), and 1 coyote (Canis latrans). The S. neurona SSURNA PCR assay and a control PCR assay using protist-specific primers were applied to all sporocyst DNA samples. All sporocyst DNA samples tested positive on the control assay. The SSURNA PCR assay yielded a 484-bp product only when applied to opossum samples. The SSURNA gene of both opossum sporocyst samples was sequenced to determine its relationship to the S. neurona SSURNA gene. The sequence had 99.89% similarity with S. neurona. This suggests that opossums are the definitive host of S. neurona.

PMID: 8544064 [PubMed - indexed for MEDLINE]

1: J Parasitol. 1995 Dec;81(6):930-5.

Sarcocystis falcatula from passerine and psittacine birds: synonymy with Sarcocystis neurona, agent of equine protozoal myeloencephalitis.

Dame JB, MacKay RJ, Yowell CA, Cutler TJ, Marsh A, Greiner EC.

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Equine protozoal myeloencephalitis (EPM) is a neurologic disease of horses caused by Sarcocystis neurona. The horse is a dead-end host for S. neurona and the definitive and intermediate hosts have not previously been identified. We hypothesized that S. neurona is actually Sarcocystis falcatula, a parasite that cycles in nature between Virginia opossums (Didelphis virginiana) and any of a variety of avian intermediate hosts. We extracted DNA from S. falcatula sarcocysts in the muscle of a brown-headed cowbird (Molothrus ater) and from schizonts in a fixed specimen of lung from a Moluccan cockatoo (Cacatua moluccensis). Three segments of the small subunit ribosomal RNA (SSURNA) gene, containing a total of 742 nucleotides, were amplified by the polymerase chain reaction, sequenced, and compared with the SSURNA sequence from two isolates of S. neurona. The S. falcatula sequence was identical to the sequence of the S. neurona isolate UCD-1 and differed in only 3 positions from isolate SN5. Recent evidence, also based on SSURNA sequences, implicates the opossum as the definitive host of S. neurona. Based on the SSURNA gene sequences S. falcatula and S. neurona are synonymous, thus the parasite cycles between opossums and birds maintaining a reservoir of the organism from which horses can be infected.

PMID: 8544067 [PubMed - indexed for MEDLINE]

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Veterinary Parasitology
Volume 92, Issue 2 , 20 September 2000, Pages 119-128

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Neospora hughesi: experimental infections in mice, gerbils, and dogs

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Abstract

Neospora hughesi is a recently described cause of equine protozoal myeloencephalitis (EPM). A rodent model for pathogenicity would facilitate development of therapies to be used in horses. In the present study, we examined the susceptibility of BALB/c γ-interferon gene knockout (γ-INFKO), BALB/c, CD-1, and C57BL/6 strains of mice and gerbils to infection with tachyzoites of the Nh-A1 strain of *N. hughesi* isolated from a horse from AL, USA. Only the γ-IFNKO mice developed severe clinical disease following infection with *N. hughesi* and died 19–25 days after infection and exhibited severe cardiac lesions. In contrast, experimental infection of γ-INFKO mice with tachyzoites of the NC-1 or NC-Liverpool strains of *Neospora caninum* resulted in deaths 8–10 days after infection. The most severe lesions were in the livers, spleens, and lungs of these mice. Gerbils inoculated with *N. hughesi* did not develop clinical disease, had few microscopic lesions, but did seroconvert. Two dogs fed the brains of mice, shown to contain *N. hughesi* tissue stages by cell culture and γ-IFNKO mouse bioassay, did not shed *N. caninum*-like oocysts over a 23 days observation period. The marked difference in pathogenicity between the two species of *Neospora* in γ-IFNKO mice, and lack of oocyst excretion by dogs fed *N. hughesi* infected mice provide additional evidence that the species distinction between *N. caninum* and *N. hughesi* is valid.

Author Keywords: *Neospora hughesi*; *Neospora caninum*; Mouse; Gerbil; Dog; Horse; Model.

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1: J Parasitol. 1999 Oct;85(5):979-81. Related Articles, Links

Simplified technique for isolation, excystation, and culture of Sarcocystis species from opossums.

Murphy AJ, Mansfield LS.

Animal Health Diagnostic Laboratory, Michigan State University, East Lansing 48824, USA.

Sarcocystis neurona is a protozoan parasite that causes a neurological disease in horses called equine protozoal myeloencephalitis. The route of transmission is speculated to be by fecal-oral transfer of sporocysts shed from opossums. Controversy exists regarding both the natural life cycle for this parasite as well as the species identity of opossum Sarcocystis. To provide stage-specific material for species comparison, 27 opossums from southern Michigan were screened for Sarcocystis spp. sporocysts. Seven opossums were positive for Sarcocystis sporocysts by fecal flotation. A simplified, effective technique for isolation, excystation, and culture of opossum Sarcocystis sp. from mucosal scrapings was developed. All 7 Sarcocystis sp. isolates were successfully cultured to grow long term in equine dermal cells to the merozoite stage. Merozoites were observed between 5 and 15 days after inoculation. In conclusion, opossums shed Sarcocystis sp. sporocysts that may be manipulated to excyst and grow in vitro in equine dermal cell lines to the merozoite stage using the simplified technique described.

PMID: 10577742 [PubMed - indexed for MEDLINE]

Vet Clin North Am Equine Pract. 1997 Apr;13(1):79-96. Related Articles, Links

Equine protozoal myeloencephalitis.

MacKay RJ.

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Equine protozoal myeloencephalitis (EPM) is a common neurologic disease of horses in the Americas. Horses with EPM most commonly have abnormalities of gait, but they also may present with signs of brain disease. The disease ranges in severity from mild lameness to sudden recumbency, and clinical signs usually are progressive. A causative agent, Sarcocystis neurona, has been isolated from affected horses, and serologic surveys suggest that approximately 50% of horses in the United States have been exposed. EPM is considered a treatable disease, although the response to antimicrobial treatment often is incomplete. This article highlights new information about the life cycle of S. neurona and reviews the literature regarding diagnosis, clinical signs, and treatment of the disease.

Publication Types:
- Review
- Review, Tutorial